CORRELATION OF CRP LEVEL WITH GLYCEMIC CONTROL IN DIABETIC FOOT PATIENTS AND ITS SEQUELAE

Dissertation Submitted to

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M.S. GENERAL SURGERY

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CERTIFICATE

This is to certify that the dissertation titled **" CORRELATION OF CRP LEVEL WITH GLYCEMIC CONTROL IN DIABETIC FOOT PATIENTS AND ITS SEQUELAE "** of **Dr. A.ANAND** in partial fulfillment of the requirements for **M.S. Branch – I (General Surgery)** Examination of the Tamilnadu Dr. M.G.R. Medical University to beheld in APRIL 2013. The period of study was from November 2011 to November 2012.

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DECLARATION

I, **Dr.A.ANAND** solemnly declare that dissertation titled, "**CORRELATION OF CRP LEVEL WITH GLYCEMIC CONTROL IN DIABETIC FOOT PATIENTS AND ITS SEQUELAE "** is a bonafide work done by me at Govt.Stanley Medical College & Hospital during 2011-2012 under theguidance and supervision of my Unit Chief

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The dissertation is submitted to Tamilnadu Dr. M.G.R. MedicalUniversity, towards partial fulfillment of requirement for the award of **M.S. Degree (Branch – I) in General Surgery.**

(Dr.A.ANAND)

Place : Chennai.

Date :

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CONTENTS

INTRODUCTION

The term **diabetes** was coined by **Aretaeus** of Cappadocia.it was derived from the greek word "**diabainein**". Diabetes means one that straddles. Diabetes was recorded first in English as Diabete around 1425.

In 1675, the word mellitus was added by Thomas willis. Matthew Dobson in 1776,confirmed the excess sugar in urine and blood.

Sushrutha named diabetes as **Madumeha**(sweet urine disease) in 6^{th} century. Avicenna Persian (980–1037) described diabetic gangrene.

The role of pancreas in diabetes was first by Joseph von Mering and Minkowski. 1910 sir Edward albert Sharpey Shafer suggested that diabetic people were deficient in insulin.

Banting and **Best** purified insulin from bovine pancreas for which he was awarded noble price. Banting is honoured by worlds diabetes day which is held on his birthday, November, 14.

Amputations surgical principals was first given by **Ambrose Pare.**

AIMS AND OBJECTIVES

The purpose of the present study is

To analyze the level of CRP and adequacy of glycemic control in type 2 DM with diabetic foot on treatment.

To correlate the level of CRP with sequelae of diabetic foot.

REVIEW OF LITERATURE

Lin C W et al, after analysing 90 diabetic foot patients and concluded that reduced c reactive protein level (<50mg/l) indicates good prognosis in diabetic foot patients.

1. **Masayo Fukuhara et al**,studied 195 residents and concluded good glycemic control in elderly reduces systemic inflammation that contributes to atherosclerosis in this study HbA1c was used as glycemic index CRP used as marker for systemic inflammation.

2. **Baris A kinci**, of DokuzEylul university hospital(2003-2008) observed 165 ulcer patients and concluded that post treatment CRP values were strongly related to amputations.

3. **Dana.E.Kinget al**, (NHANES III survey), 1988-1994 in his study concluded that CRP concentrations increased with raised HBA1c level and shows correlation between glycemic control and systemic inflammation in patients with proven diabetes.

4. **VOlaco Et al,** Heinrich-Heine university, Germany studied 98 diabetic foot patients with ischemic lesions and published that

high CRP levels are predictive of non healing ulcer and amputation.

5. **Anthonia.O.Oghera12**, Langlos state university teaching hospital, Lagos state , studied 200 type 2 diabetes patients and published that elevated CRP and lipoprotein a was observed in diabetics than in non diabetics.

6. **Christian Weigelt et al, erman** diabetic clinic (2003-2005) studied 125 patients with diabetic foot and his study revealed that diabetic foot ulcers and its complications are associated with elevated c reactive protein levels.

7. **Farah Jabeen Et al,** studied 51 patients with type 2 diabetes and concluded that elevated CRP levels and alteration in platelet morphology helps to identify diabetic patients who are at risk of developing complications.

   Pak J med sci, jan- mar 2012 vol.29, no. 1

8. **Mark.B.Pepys et al**, a study in Royal free and university medical school UK (2003) revealed increased CRP level can predict peripheral vascular events in diabetic patients.

9. **Wong Et al,** (DIGAMI study (8), 1997 proved a positive correlation between c reactive protein level and blood sugar level Editorial (Wong et al.., p 2971).

10. **Bejamin .A. Lipsky et al,** evaluated 402 diabetic foot patients and concluded that severe wound grade ,elevated WBC count and increased CRP can predict unfavourable outcome in diabetic foot patients.

11. **KaushikBhowmick et al** ,R.L.Jalappa hospital, Kolar ., Karnataka.(July 2005 –Dec 2005) studied patients with Type 2 DM and concluded positive correlation between CRP, microalbuminuria in Type 2 DM with poor glycemic control and normal CRP levels in adequately controlled patients.

    Indian Journal of Biochemistry.2001/22(2) 53-59.

12. **Mohammed R. Ahmed**, Zagazig university, Egypt published that in diabetic foot patients c reactive protein and MIF helps in differentiating infected from non infected diabetic foot.

13. **Debra. A.Schaumberg et al** ‘s study ,diabetes control and complications trial demonstrated intense glycemic control in

diabetic patients reduces C reactive protein , TNF –R. Elevated CRP levels were associated with risk of atherosclerosis.

14. **Lee Et al,** study on diabetic foot patients showed that elevated CRP values are more useful in predicting infection when compared to WBC count and ESR.

15. **SabiullahAmanullah ,** Al Arab Medical university ,Benghazi , Libya, compared association of CRP in diabetic and non diabetic patients and that study revealed a strong correlation between CRP and glycemic control in diabetic individuals.

    Jordan Journal Of Biological Sciences,

    Volume 3, number 1, January 2010.

16. **Upchurch Et al,** compared CRP level in diabetic foot ulcer patients and diabetic patients without ulcer and concluded CRP level in foot ulcer patients.

17. **Pradhah Et al,2001**; Haffeiner(2003); Hanley Et al.(2004) studied CRP levels in diabetic patients and concluded that CRP level elevated in diabetic patients and can be used as biomarker for inflammation.

18. **Jeandrot Et al,** study proved CRP has high sensitivity and speficity in predicting infection in diabetic foot patients.

19. **Rodriguez and Guerrero (1993**) ,studied patients with type 2 diabetes patients and concluded that decreased CRP levels were observed in patients with low HBA1c and a positive correlation between CRP and glycemic control.

20. **King DE et al,** study proved elevated CRP level correlates with raised HBA1c levels, showing association between glycemic control and systemic inflammation.

    Department of family medicine, medical university of south Carolina , Charleston 29425,USA.

21. **Li et al.2004and Aronson et.al** 2004 study in diabetic patients showed positive correlation between CRP and HBA1c level and independent association of CRP with fasting plasma glucose.

22. **Scholin A et al** studied 97 diabetic patients and concluded that elevated CRP levels has positive correlation with poor glycemic control.

Department of medical sciences, Section of Internal Medicine, Uppsula university hospital, Sweden.

23. **Li cz et al (2004)** study showed positive correlation between fasting blood sugar level with c reactive protein level.

DIABETES MELLITUS

Diabetes and its complications is one of the most common cause of death. Prevalence of Diabetes worldwide is around 252 million. prevalance of diabetes in India is 42 million and age of distribution is middle and elderly people.

There is 20% risk of developing diabetic ulcer in diabetic patients over a period of time.

DIABETES MELLITUS CLASSIFICATION:

Type 1 IDDM (insulin dependent diabetes mellitus)

Type 2 NIDDM

Other types: genetic

Infections

Drugs/chemicals

Endocrinopathies

Gestational DM

DIABETIC FOOT

Diabetic foot is the common complication in lower extremity. 13 – 18% (percent)of patients will develop diabetic ulcer in foot over a period of time.

Autonomic neuropathy, poor glycemic control, high pressure in soles are the factors which contributes to development of ulcer and one of the cause of non traumatic amputation of lower limb.

In terms of level of amputation toe amputation is the most common. Rate of amputation is 15 to 40 times high in diabetic patients when compared to normal.

ULCER RISK:

Single most common factor which determines amputation in lower limb is foot ulceration.

Risk factors which leads to foot ulceration involves polyneuropathy which includes

Autonomic

Sensory

Motor neuropathy,

Trivial trauma

Atherosclerosis of involved vessels.

Approximately 50% of foot ulcerations are purely neuropathic, remaining have neuropathic and vascular components. Out of which sensory neuropathy is the leading factor which contributes to diabetic foot ulceration.

Motor neuropathy leads to muscle wasting and foot deformities. Additionally autonomic neuropathy Results in dry skin which cause fissures and crack causing portal for entry of bacteria.

Common deformities:

Hallus valgus

Prominent metatarsal head

Charcot joint

Hammer toe deformity.

Peripheral arterial disease also leads to foot ulceration.

Mechanism being

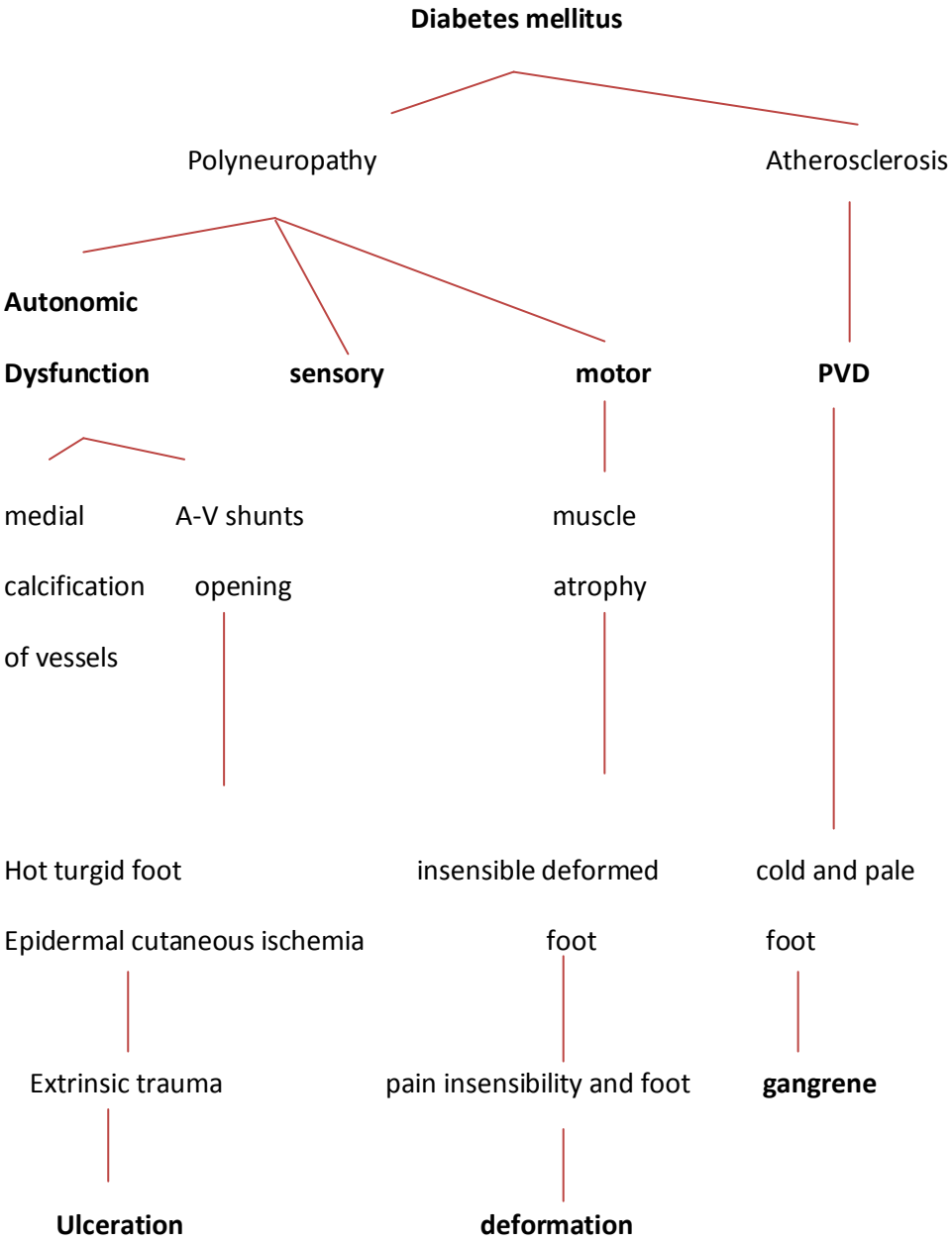
Due to arterial insufficiency

prolonged healing time

Lack of oxygen delivery to tissues.

Delivery of antibiotic to infected site is impaired.

Due to longstanding diabetes glycosylation of collagen leading to thickening of ligaments and capsule**(CHIEROARTHROPATHY**) which subsequently reduces mobility of joint, high plantar pressure and development of ulceration.


Diabetes mellitus
Polyneuropathy
Atherosclerosis
Autonomic
Dysfunction
sensory
motor
PVD
medial
calcification
of vessels
A-V shunts
opening
muscle
atrophy
Hot turgid foot
Epidermal cutaneous ischemia
insensible deformed
foot
cold and pale
foot
Extrinsic trauma
pain insensibility and foot
gangrene
Ulceration
deformation

DIABETIC FOOT INFECTIONS:

Diabetic foot infections are typically polymicrobial.

Hyperglycemia

Atherosclerosis

Neuropathy

Immunocompromised status are contributing factors which leads to foot infections in diabetes.

Due to impaired immune response and ischemia infection control in diabetes is poor with antibiotics. Poor infection control leads to cellulitis cause irreversible damage to tissues.

Diabetic ulcer classification system:

Wagner classification system

Grade	**Lesion**
0	cellulitis or deformity, no lesion
1	Superficial diabetic ulcer
2	Ulcer extension Involves ligaments tendons joint capsule or fascia No abcess or osteomyelitis
3	Deep ulcer with osteomyelitis or abcess formation
4	Gangrene to portion of fore foot
5	Extensive gangrene of foot

University of texas classification:

Stage	0	1	2	3
A	completely epithelialised wound	superficial wound	Depth upto tendon or capsule	Involving bone or joint
B	presence of infection	Infection	Infection	Infection
C	presence of Ischemia	Ischemia	Ischemia	Ischemia
D	presence of both Infection and Ischemia	Infection and Ischemia	Infection and ischemia	Infection and ischemia

AMPUTATION IN DIABETES:

The incidence of amputation is 15 to 40 times greater in diabetes when compared to non- diabetes.

And it serves as the marker for disease severity. Impairment of blood supply due to atherosclerosis .May also be an independent factor causing amputation and predisposes to gangrene.

Infection and chronic hyperglycemia has also been implicated as risk factor leading to amputation.

Infection leads to sepsis, soft tissue destruction, abcess formation and osteomyelitis.

Chronic hyperglycemia causes microangiopathy, glycosylation of collagen and affects host phagocytosis mechanism.

Both these factors will ultimately leads to amputation. Past history of amputation and ulcer in the lower extremity are major determinant factors for subsequent amputation.

ASSESSMENT OF DIABETIC FOOT ULCER:

Complete history and physical examination

Duration

Glycemic status

Existence of other comorbid conditions

FOOT SPECIFIC

Foot deformity

Callus formation

Previous infection

Surgery.

Laboratory screening:

Fasting and post prandial blood sugar values

To calculate insulin dose

To assess glucose level

Complete blood count and RFT

To look for changes due to diabetic nephropathy.

HBA1c

To look for long term glycemic control

Erythrocyte sedimentation rate

C reactive protein

To look for severity of infections

Alkaline phosphatase :

will be elevated in diabetes

Wound culture

To start the patient on appropriate antibiotics

Serum lipid profile:

It tells about vascular risk(atherosclerosis)

Urinalysis

Ketones

To rule out ketoacidosis

Dietary habits

IMAGING STUDIES:

Plain radiograph :

Detect bone changes like

1. osteolysis
2. osteomyelitis
3. fractures
4. dislocations.

Technetium-99 methylene diphosphonate:

Predict presence of osteomyelitis.

Increase uptake is seen in fracture, infections and neuropathy.

Technetium-99 sulfur colloid:

Actively functioning bone marrow will show increased uptake, as in osteomyelitis

Differentiates osteomyelitis and neuropathic arthropathy.

Indium 111 scans:

Leucocytes are selectively labled in indium scan

Specific for osteomyelitis.

CTscan:

For joint and bony changes that are not seen in x rays.

Subluxation can be visualized.

Biopsy:

Correlates best with the duration of diabetes

Neuropathies will not occur in the initial period of onset of diabetes

The following nerve fibre changes are observed in histopathology

1. myelination changes
2. nervefibres density will be reduced
3. degeneration of axons
4. degeneration of pericyte in interstitium
5. reduplication of basement membrane(neurilemma).

NEUROLOGICAL EVALUATION:

Semmes Weinstein monofilament wire for assessing sensory function.

Patient not able to feel- loss of sensation present Biothesiometer for assessing vibration perception.

>25volts significant neuropathy.

Nerve conduction studies: In diagnosing peripheral neuropathy.

Pin prick

VASCULAR EVALUATION:

Palpation of pulses

NON palpable pulses :

Doppler segmental arterial pressure

Toe blood pressure:

Normal more than 60mm of Hg.

Less than 40 critical limb ischemia.

Doppler USG: Helps in planning further management

Shows level of occlusion in macrovascular disease

Decide level of amputation.

Laser Doppler velocimetry and measuring skin perfusion

Pressure: assess flow in capillaries and superficial arterioles

Predicts critical limb ischemia.

Toe brachial index

More than 0.7 – normal.

Less than 0.64- arterial disease.

Arteriography: assess the level of arterial occlusion

To visualize collaterals

Done by seldingers technique.

To visualize distal run off

For revascularization procedures

MR angiography and CT angiogram: evaluates distal arterial perfusion

Ankle brachial index

Interpretation of ABI

Resting ABI	**severity of obstruction**
0.91-1.30	normal
0.70-0.90	mild
0.40-0.69	moderate
<0.40	severe

Indication for vascular intervention:

ABI < 0.7

Toe blood pressure< 40 mm of hg

Transcutaneous oxygen tension < 30 mm of hg

These factors are associated with poor healing.

ASSESSING PLANTAR FOOT PRESSURE:

THE HARRIS MAT(computerized): can identify areas vulnerable to ulceration

Measures plantar foot pressure

PRESSURE STAT(non computerized): uses pressure sensitive sheets

Used as screening tool to identify areas easily vulnerable to ulceration.

Foot ulcer evaluation:

Assess

Skin changes around the ulcer

Ulcer characteristics- size site shape

Condition of ulcer- wound edges and bed

Presence of necrosis, pain, cellulitis and gangrene

RISK CATEGORIZATION SYSTEM:

Category	Risk	Evaluation
0	Normal	every year
1	Presence of peripheral Neuropathy	every 6 months
2	Presence of peripheral Neuropathy, PAD or Deformity	every 4 months
3	Amputations Past h/o ulcer	monthly to quaterly

PAINFUL NEUROPATHY MANAGEMENT:

General measures:

1. Control blood sugar
2. Stop alcohol
3. Vitamin b12 supplementation
4. Educate the patient

Medical measures:

Tricyclic anti-depressants

Anti –Convulsants

1. Sodium valproate.

2. Phenytoin sodium.

3. Carbamazepine.

DRESSINGS IN DIABETES:

Hydrocolloid dressing:

Contains carboxy-methyl- cellulose and gelatin.

Often combined with adhesives and elastomers.

Wound debridement occurs by autolysis.

Frequency of dressings can be reduced until 7 days.

Disadvantages:

Hypergranulation on long term use

Hydrogel dressing:

Hydrate wounds and debridement occurs by autolysis.

Can be useful in painful and burning wounds.

Thick gels: PURILON

INTRASITE

Thin gels: SOLUGEL

SULUSITE

Can be kept in wound for 3 days.

Alginate dressing:

Synthesised from sea weed.

Contains calcium ions – biodegradable.

Needs active exudation from the wound for it to function.

Contraindicated in dry wounds.

Foam dressing:

Used in exudative wound due to its absorbable property

Provides thermal protection

Cannot be used in the presence of infection

Can be impregnated with silver and thus acts as bactericidal dressing

Iodine preparations:

Antiseptic.

Bactericidal action.

Used in infected wounds.

Usually combined with systemic antibiotics.

Prevents skin excoriation in exudating ulcers.

Especially used for cavitary wounds.

TREATMENT OF DIABETIC FOOT:

Wound bed preparation:

It has been proved that diabetic wound will not heal in the presence of debris, non viable tissues and bacterial colonisation. Wound debridement became integral part in the management of diabetic foot.

Debridement

Removes all necrotic tissues and callus.

Reduces pressure.

Helps in evaluating wound bed.

Reduces bacterial burden.

Facilitates drainage of abcess.

Adequate debridement must be done before applying topical healing agents and wound closure.

Various types of debridement:

Surgical

Enzymatic

Autolytic

Mechanical

Biological

Out of which only surgical debridement has been proven to be effective.

Surgical debridement is done until healthy soft tissues active bleeding and bones are encountered.

Main aim of debridement is to convert chronic non healing ulcer into acute healing ulcer.Any deep abcess associated with diabetic foot should be drained. If osteomyelitis is encountered amputation should be

done. Necrotic tissues should be excised and debrided regularly. Frequent maintainence debridement is mostly required.

Enzymatic debridement:

Application of synthetic proteolyic enzymes for wound debridement.

Eg.bacterial collagenase, papain, trypsin, fibrinolysin.

Autolytic debridement:

Occurs in moist healthy environment.When blood supply is maintained.

Mechanical debridement:

This includes wet to dry dressing

High pressure irrigation

Pulsed lavage and hydrotherapy.

Biological therapy:

Here proteolytic enzymes secreted by maggots is used that liquefies necrotic.

Tissues to get nourishment for the development during their life cycle.

Infection and inflammation control:

Infection may be local involving

soft tissue,

systemic or

ascending infection.

As host immune system is compromised signs of inflammation will be absent. In such case elevated blood sugar value serves as marker for infection. Obtain culture and sensitivity report before starting antibiotics.

ADVANCED MODALITIES IN WOUND CARE:

Growth factor therapy

Genetically engineered platelet derived growth factor

Becaplermgel(REGRANE x tm)

Works by stimulating chemotaxis.

Autologous platelet rich plasma:

Stimulates platelet alpha granules to release various growth factors.

Bioengineered tissues:

Currently two products have been approved

1. Apligraft
2. Dermagraft

Bilayered skin substitutes:

Bilayered cultured composite skin.

Extracellular matrices:

From devitalized tissues

Dermal regeneration template (integra tm)

Allogenic dermal matrix (alloderm)

Matrix of human dermal fibroblast.

Adjunctive modalities:

1. Regerative tissue matrix

2. Hyperbaric oxygen therapy

3. Ultrasonic therapy-MIST therapy tm system

Uses fine saline spray that allows USG administered directly to wound.

4. **negative pressure wound therapy**

Vacccum assisted closure device

5. **electrical stimulation.**

PRESSURE RELIEF /OFF- LOADING:

Totally non weight bearing- bed rest, crutches, foot casts or boot.

Rocker bottom sole with removable walking brace.

Custom walking brace.

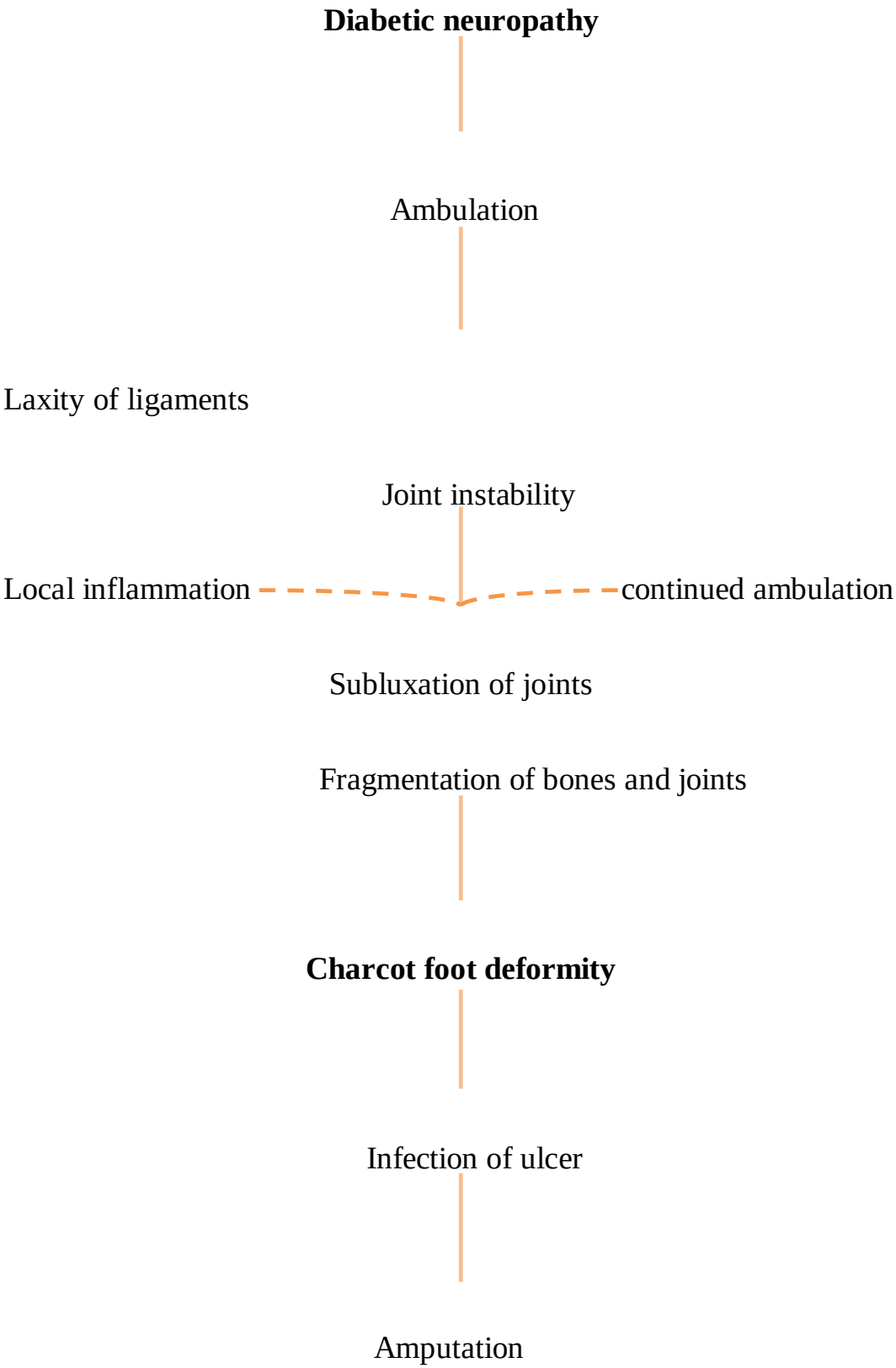
Wedge shoes/half shoes.

Tendon bearing braces- patella.

Assistive device- crutches cane walker.

Accommodative dressing.

MANAGEMENT OF CHARCOT FOOT:



Charcot arthropathy classification:

Eichenholtz classification system:

Developmental active stage- active stage

Coalescent – quiescent or reparative.

Reconstructive – quiescent or reparative stage.

Management of acute neuroarthropathy:

Main stay of treatment is Immobilization and stress reduction.

Initial offloading:

Non weight bearing- uses of crutches.

Short leg plaster.

Fibre glass non weight bearing cast.

Pneumatic walking.

Removable cast walker.

Post acute phase of treatment:

Following reduction in temperature and edema

Protective weight bearing.

Total contact cast: fixed ankle walker.

Bivalve cast total contact prosthetic walker patellar tendon bearing braces.

Moderately unstable ankles: ankle foot orthosis.

high top therapeutic shoes.

Severly unstable: patellar tendon bearing brace.

Bisphosphonate therapy acts by inhibiting bone resorption

Electrical bone growth stimulation.

Low intensity pulsed ultrasound.

Surgical management of osteoarthropathy:

Indication of surgical intervention is subluxation without osteochondral fragments.

Goal -to create **stable plantigrade foot**

Includes- Exostectomies for rocker bottom deformity.

Isolated or mid foot arthrodesis.

Hind foot fusion.

Triple arthrodesis.

Tibio calcaneal fusions.

Ankle fusion.

SURGICAL MANAGEMENT OF DIABETIC FOOT:

Recent classification based on surgery

Class 1 elective

Class 2 prophylactic

Class 3 curative

Class 4 emergent

Elective surgeries:

Goal-to relieve pain associated with deformities

All types of reconstructive surgeries fall in this category.

Eg.ankle arthrodesis, Achilles tendon lengthening.

Amputations only in severe deformity and joint instability.

Prophylactic surgery:

Goal – to prevent occurrence and recurrence of ulceration

Reconstructive procedures involving correcting tendon, bone and deformed joints fall in this category.

Curative surgery:

Goal –to promote healing of chronic non healing ulcer

Removing peak pressure areas in the foot

Removing infected bones and joints.

Eg.exostectomy, sesamoidectomy, metatarsal head removal

Joint resection partial calcanectomy.

Emergent surgery:

Goal –to stop progression of infection

Eg.removing all non viable necrotic tissues to level of bone and viable soft tissue.

Either primary closure of stump or delayed closure is done.

AMPUTATIONS CONSIDERTIONS:

Distal stump should

Easily accommodated by inert shot, prosthesis or orthotic device.

Unlikely to break due to exogenous pressure.

Should not cause dynamic or muscle imbalance.

Should heal by primary intention.

Amputation prevention programme:

1. **Foot care**

Regular self examination

Risk assessment

Early treatment and detection of early lesions

2. **Protective shoes**

Protects from injury

Well obstructed

3. **Pressure reduction**

Padded hosters

Cushioned multiple density moles

Pressure measurements

4. **Prophylactic surgery**

Correct surgical deformity

Prevent occurrence of ulcer

5. **Preventive education**

Daily inspection

Patient education

Physical education- importance of foot examination

Teaching recent concepts in diabetic foot management.

MANAGEMENT OF DIABETIC FOOT ACCORDING TO GRADE OF ULCER

Grade I diabetic ulcer:

ulcers which penetrate beyond epidermis

Indicates two risk factors involvement:

Peripheral sensory neuropathy + other risk factor.

Ulcer clinically evaluated.

Primary surgical debridement of hyper keratotic area.

Depth of defect post debridement area assessed.

Surgical debridement is the best debridement method.

Sharp debridement-

With scalpel all necrotic and dead tissues removed.

Until active bleed from the ulcer is seen.

Surgical debrimentcarried out in OR under suitable anaesthetic agent.

Pressure sites eliminated in foot by providing suitable offloading equipments.

Non-weight bearing crutches .

Due to poor compliance following this regimen is difficult.

Total contact casting.

The felted foam dressing.

It is cost effective and compliance is good.

Can be used as adjunct in the treatment of diabetic foot

Before application measure the size of wound.

Procedure:

Aperture is created in the dressing, specifically the felted foam.

Trim the edges with scissor.

Trim along the opening close to the wound.

This is to prevent "**EDGE EFFECT**".

Paint the foot with rubber cement, on both side of each felted foam.

Allow it to dry.

Felted foams are laminated.

With foam in the bottom and felt in the top.

Secure dressing with self-adherent around the foot

Create window

Which allows dressing

Direct visualization

Assess the lesion

Provide healing shoe so that the patient can wear while ambulating.

Pads kept in place for 2 weeks

Reapply if necessary.

Pros:

Frequent dressing can be done

Wound can be inspected daily.

Cons:

Foot has to be kept dry

Frequent ambulation not possible.

The ulcer is addressed daily to create favourable environment.

Systemic antibiotics to control infections according to culture reports.

Correct foot deformity surgically.

Grade II diabetic ulcer:

When patients with grade 1 lesions continue to bear weight ulceration becomes deep to involve other structures.

X ray foot has to be taken to rule out any bony changes.

Grade II lesions **should not contain any bone changes**.

Examine : for presence of sinus tract for deep structure involvement.

Diagnose osteomyelitis:

By Technetium 99 biphosphonate

Technetium 99 sulphur colloid

CT scan

Bone biopy

Management includes:

Strict bed rest

Broad-spectrum antibiotics.

Drain the foot in dependent area and pack.

This should be done in OT.

Explore and drain the sinus tract if present.

Surgical debridement of all dead tissue .

This is done thoroughly and aggressively.

Take culture from deep tissues.

Start IV antibiotics according to culture report.

Look for vascular changes by investigation.

Consult vascular surgeon after initial debridement.

Delay in surgery cause

Tissue loss

Potential limb loss

Twice daily Dressing should be done.

Frequent debridements before closure of wound.

Once granulation tissue is formed definitive treatment is done.

Or leave the wound to contract and heal by secondary intention.

Grade II diabetic ulcer takes long time to heal as the wound is deep And weight bearing might be difficult due to unhealthy scar.

If possible the wound should be closed primarily.

Treatment principles are same as of grade I ulcer

Non-weight bearing

Weekly debridement

Frequent Dressings.

Oral antibiotics.

Grade III diabetic Foot:

Grade III wound involves bone or abcess formation

Predisposing factors:

Grade II ulcers not responding to treatment

Uncontrolled bacterial infections

Puncture wounds involving bone.

Will require admission and surgical intervention .

Abcess drainage is essential .

Explore sinus tract if present drain the abcess and debride necrotic tissue.

Ray amputations may be needed in some patients control infection.

Clear the infection

Wait for the granulation to appear

Wound reconstruction .

Reconstructive surgical options:

Resection of bone,

Using flaps to reconstruct the defect,

Grafting of skin.

Due to bone involvement the patient often uses opposite limb and pressure will be transferred to adjacent foot.

Prevention:

Orthoses

Use of appropriate footwear

Podiatric visit frequently.

Change the orthoses if there are signs of breakdown.

Long term treatment goal is even distribution of plantar foot pressures over the entire foot, thereby avoiding any focal pressure.

Grade IV diabetic foot:

Risk factors which makes the management difficult includes

Presence of PVD,

Bone involvement osteomyelitis,

Presence of sepsis

Excess necrotic tissue

Management requires multidisciplinary team.

Management:

Goal is primarily to limit the tissue loss.

Arterial insufficiency if severe results in gangrene.

As end arteries are involved distal end of lowerlimb is often affected.

Inadequate perfusion

|

Poor oxygenation

|

Focal area necrosis

|

Dry gangrene.

Infection

|

Gangrene changes

|

Edema of local tissue

|

Infective vasculitis

|

wet gangrene.

Vascular insuffiency should be addressed immediately.

If possible revascularisation procedures should be done to minimize tissue loss.

Grade V diabetic foot:

Direct cause :

Arterial occlusion and failure of arterial inflow.

Amputation is the primary treatment for gangrene

Similar to grade IV lesions assess vascularity and if possible revascularisation should be done

To increase the perfusion

To prevent tissue loss and

Allow for distal level amputation.

CRP AND DIABETIC FOOT

C REACTIVE PROTEIN:

Before 1990, the value of c reactive protein assays in the diagnosis and prognosis was not clearly defined.

Later, it was found that CRP can strongly predict cardio vascular events in a value range which was previously considered normal CRP can never be used to diagnose disease, but their values can be used to interpret and it can contribute to active management of disease , just lie patient body temperature

SYNTHESIS:

CRP is synthesized by hepatocytes and its level rises to 1000 fold during inflammation CRP belongs to acute phase reactants. Spectrum of proteins the level of which may fall in response to

- Infection
- Inflammation
- Tissue damage
- Malignancy.

Cytokines stimulate production of CRP. This includes

- Interleukin 1
- Interleukin 6
- Tumour necrosis factor.

The main function of cytokine is that it acts as signal molecules which are produced by body's immune system.

Similar acute phase reactants produced by liver

- Protease inhibitors
- Members of complement system
- Carrier proteins
- Serum amyloid protein A(SAA)

The sensitivity and specificity of CRP correlates best with SAA.

Structure of CRP:

Belongs to pentraxin family arranged in cyclical manner.

Has promoters- five similar non glycosylated polypeptide non covantely bound subunits.

Contains two calcium ligands.

For CRP to bind to phosphocholine(found in the cell membrane) calcium is needed.

Due to presence of calcium in serum it helps in the binding of CRP with phosphocholine.

This clearly defines that serum calcium is the main factor which determines CRP level and CRP binding capacity with phosphocholine.

Main functions of CRP:

- Intracellular signaling.
- In turn carries Cellular reactions.

The level of serum calcium is usually low and increased level of serum calcium levels is observed in

Cell damage

Apoptosis

CRP

Binds to phosphocholine

In damaged and dead cells

Increased lyso-phospholipid

Lyses plasma membrane

Binding of CRP with dead cells

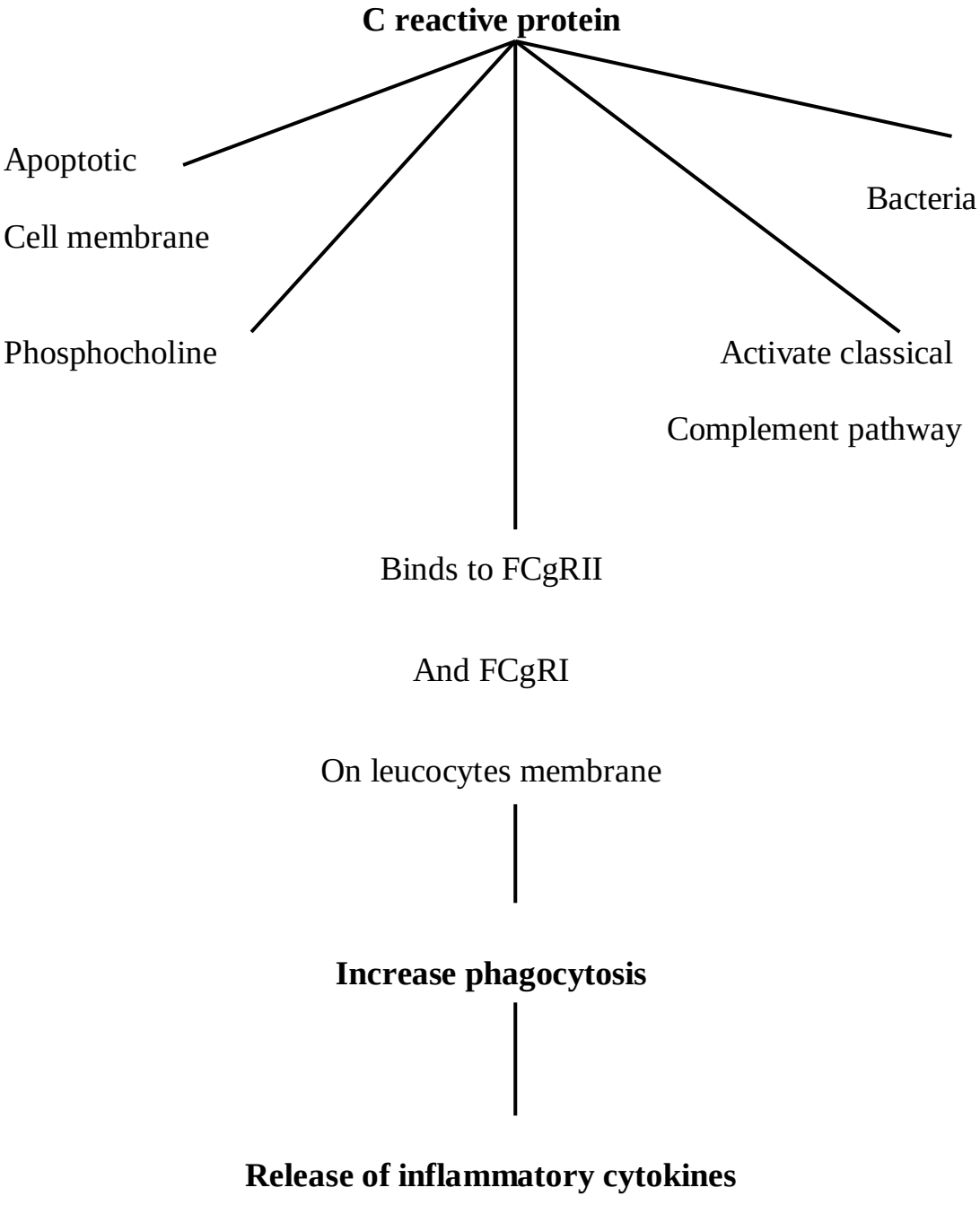
Physiological properties of CRP:

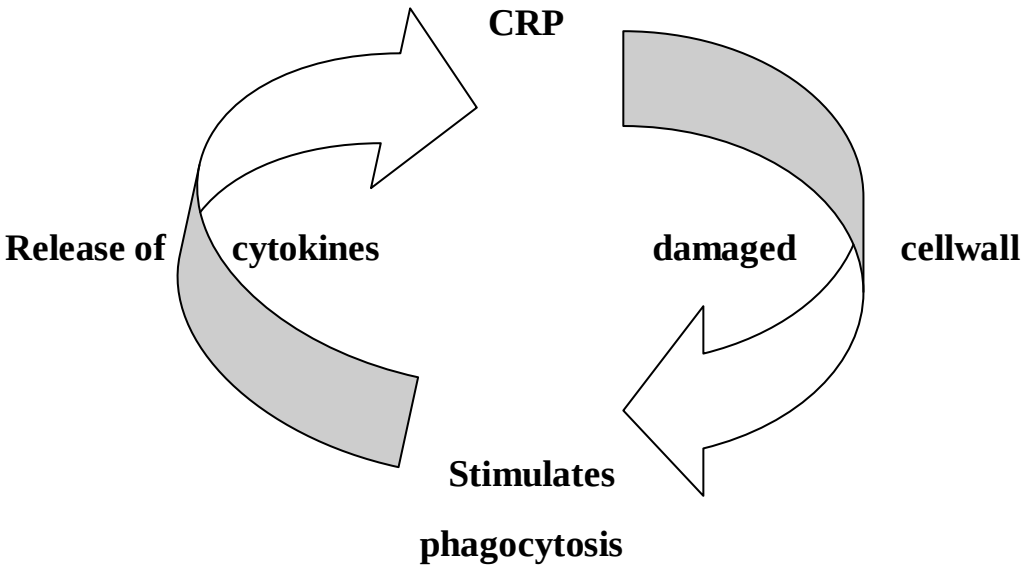
CRP activates complement system

classic complement pathway

Normally phosphocholine is exposed in bacterial cell wall but not in human cell.

When damaged by complement system, phosphocholine becomes exposed CRP binds to damaged cell membrane.





Serum concentrations:

Following single stimuli around 5mg/dl of CRP will be produced.

Peak value – 48 hours

Half life – 19 hours

Mild inflammation < 40 mg / dl

Severe bacterial infections 40 – 200mg /dl

No diurnal variation

Hepato cellular failure results in decreased production of CRP

By measuring CRP

Screen for organic disease

Monitor treatment response in inflammation and infection.

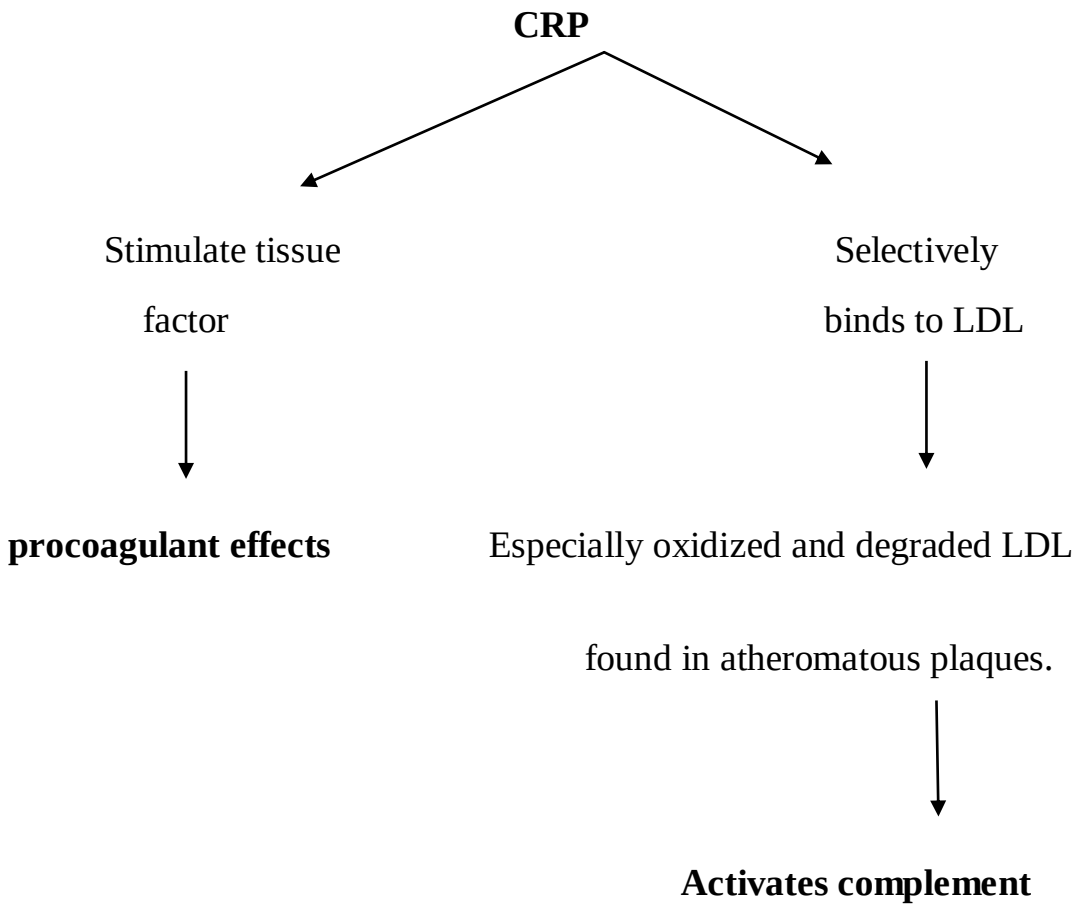
CRP and atherosclerosis:

Crp can in directly measure atherosclerosis.

Any chronic inflammation is pro-atherogenic, even if non vascular.

There is a positive correlation between CRP and atheromatous events.

Elevated CRP levels are associated with metabolic syndrome and diabetes mellitus.


CRP
Stimulate tissue factor
Selectively binds to LDL
procoagulant effects
Especially oxidized and degraded LDL found in atheromatous plaques.
Activates complement

MATERIALS AND METHODS

Setting	**:**	Department of General Surgery, Government Stanley Hospital, Chennai
Study design	**:**	PROSPECTIVE OBSERVATIONAL STUDY
Study period	**:**	1 years
Materials	**:**	Approx. 100 patients
Inclusion Criteria	**:**	All type 2 DM patients on treatment and diabetic foot.
Exclusion criteria	**:**	patients with clinical or biochemical evidence of sepsis other than diabetic foot. Patients with active auto immune disease(RA, SLE,systemic sclerosis)

All patients admitted in SMC-GS ward with diabetes and diabetic foot during JAN 2012 – NOV 2012 are included in this study. Thorough history and clinical examination done.

Admission sugar values, CRP values are obtained. Blood sugar and CRP estimation done by standard method. Grading of diabetic foot is done as per wagner classification.

As per the standard protocol all patients treated with medical and surgical care as available in institution. Throughout the admission serial measurements of blood sugar are done and documented. Weekly CRP values are obtained till discharge.

Using statistical analysis sugar values and CRP values are correlated with the grade of diabetic foot and treatment outcome.

The study is done after the patients sign the informed consent form.

OBSERVATION AND RESULTS

From January 2012 – November2012 , a total of 100 patients who was admitted with diabetic foot in the Department Of General Surgery ,Government Stanley Hospital was studied.

After admission routine blood investigations radiological evaluation. Wound swab culture and sensivity were done.

Blood sugar values both fasting and post prandial and c reactive protein levels were checked in all patients.

Both oral and intravenous antibiotics were prescribed according to the pus culture and sensitivity report.

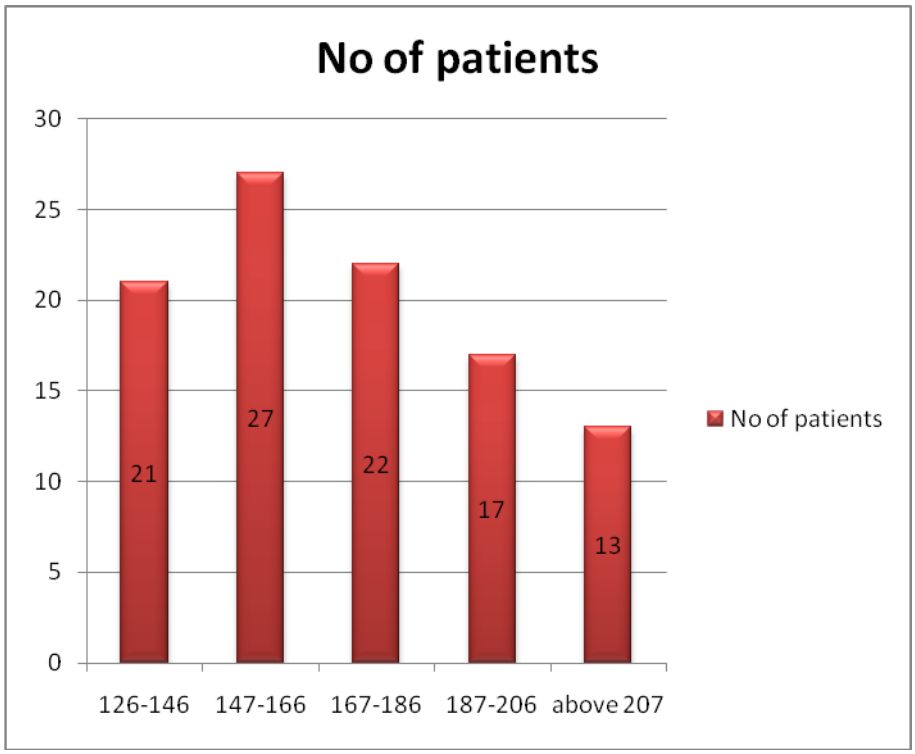
Initial through surgical debridement was done

Daily dressings were done.

The fasting blood sugar value ranges from 127 to 225 .minimum value is presentation 127 and the maximum value 225.patients are categorized into five groups to find the distribution and they are shown in the table below.

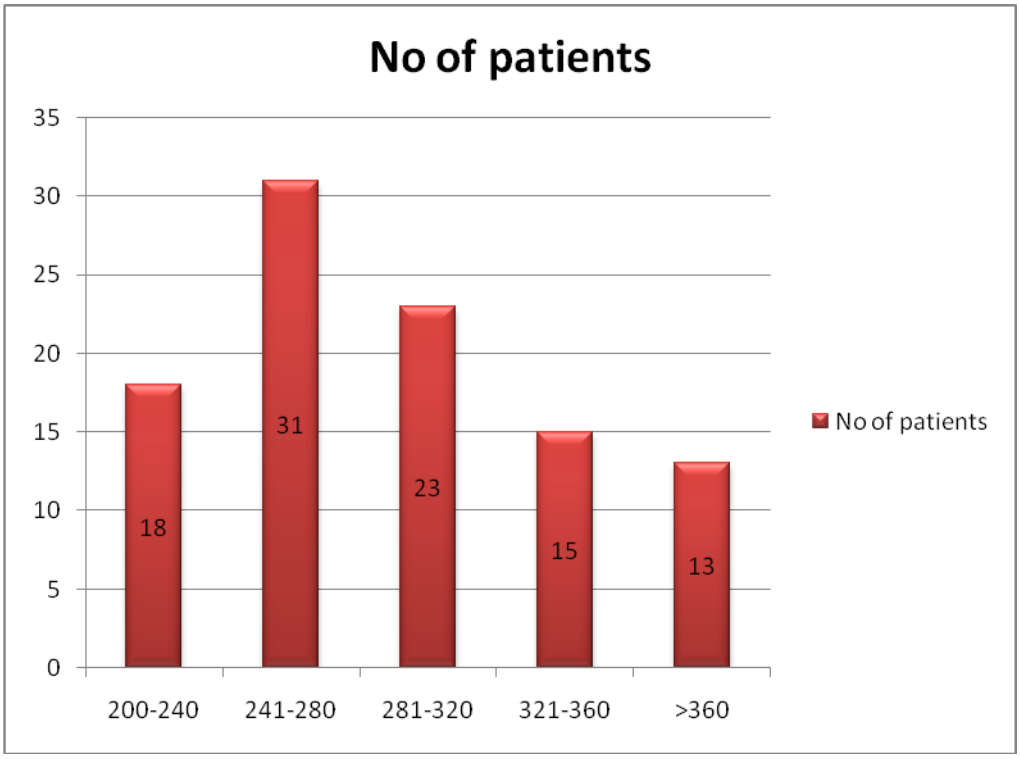
FASTING BLOOD SUGAR	NO OF PERSONS	PERCENTAGE
126-146	21	21%
147-166	27	27%
167-186	22	22%
187-206	17	17%
>206	13	13%

DISTRIBUTION OF PATEINTS IN RELATION TO FASTING SUGAR.



POST PRANDIAL BLOOD SUGAR	NO OF PERSONS	PERCENTAGE
200-240	18	18%
241-280	31	31%
281-320	23	23%
321-360	15	15%
>360	13	23%

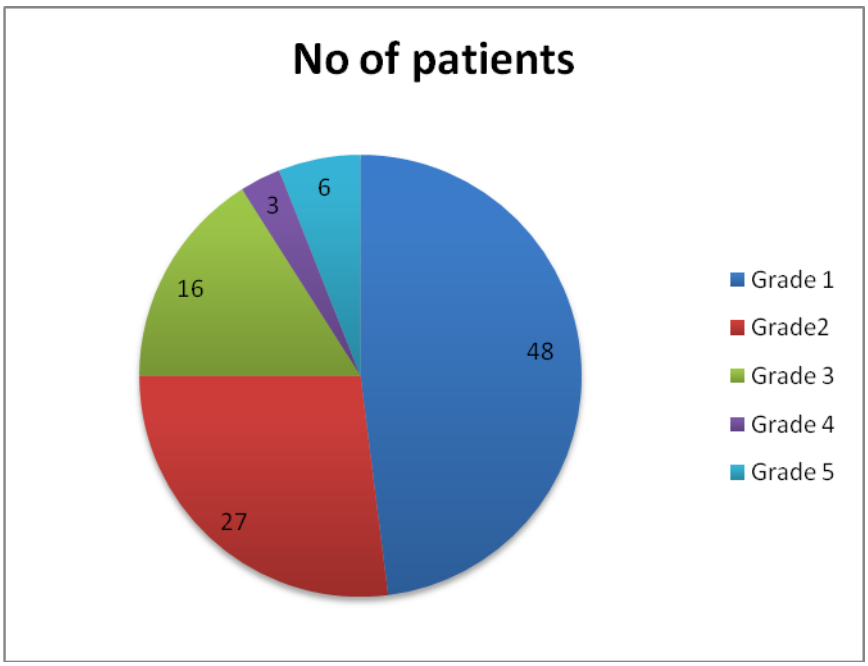
Similarly patients are classified into five groups based on the post prandial blood sugar values. Lowest value 203 and highest sugar level 394. The distributions are shown in the table above.



Based on wagnersgrading which describes the depth of wound patients are Categorized. And the table below shows the distribution of patients according to the severity of wound at presentation.

WAGNER'S GRADING	NO OF PATIENTS	PERCENTAGE
GRADE 1	48	48%
GRADE 2	27	27%
GRADE 3	16	16%
GRADE 4	3	3%
GRADE 5	6	6%

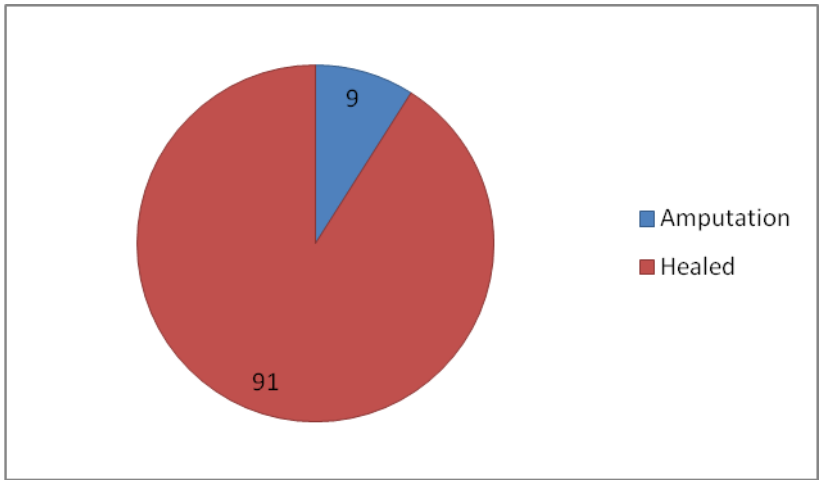
DISTRIBUTION OF PATIENTS ACCORDING TO WAGNERS GRADING



Outcome	Number
Amputated	9
Healed	91

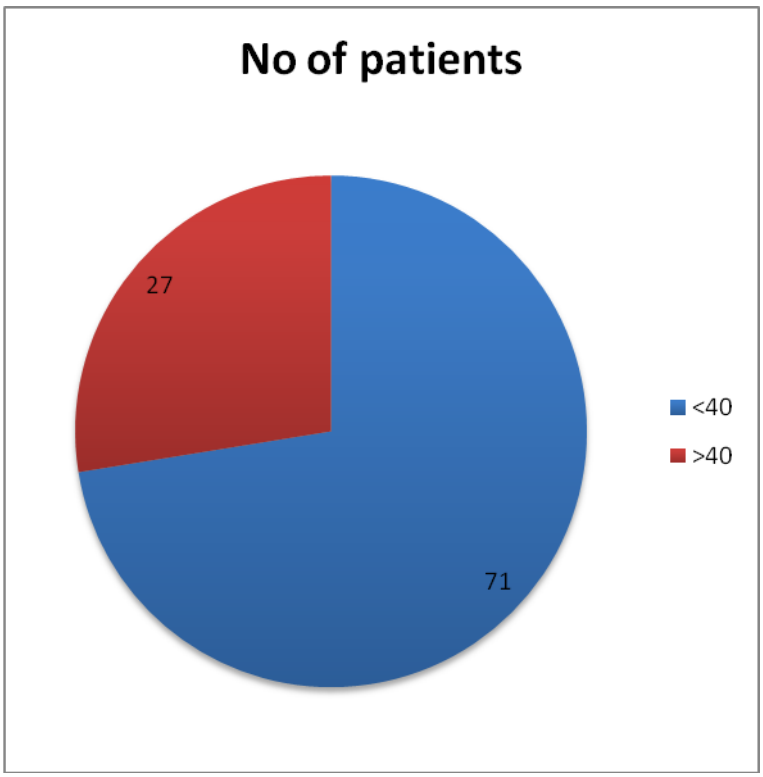
This chart implies the outcome in diabetic foot patients. Out of 100 patients 9 got amputated and in 91 patients wound healed without complications.

OUTCOME

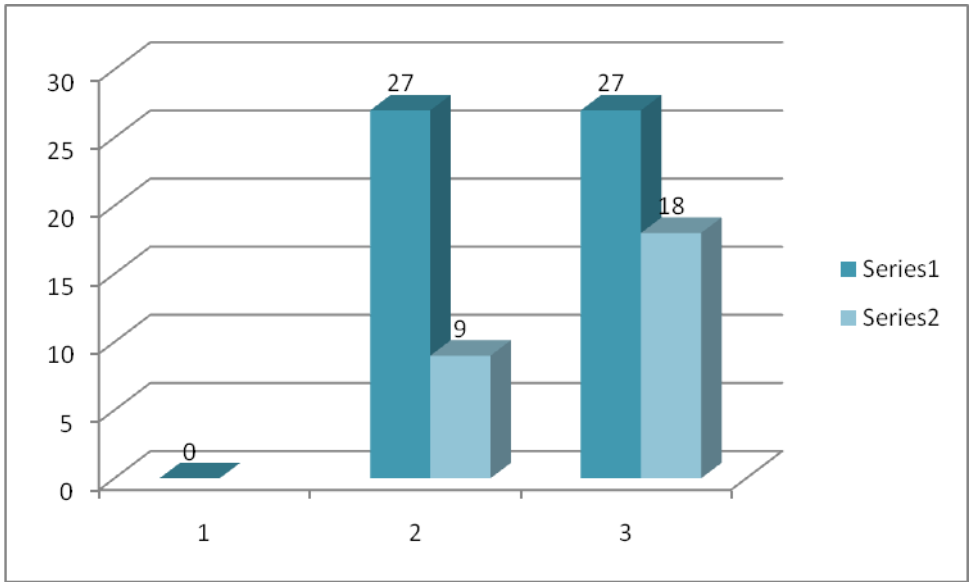


CRP VALUE	NO OF PATIENTS
< 40	73
>40	27

This chart depicts the number of patients whose CRP value was greater than and less than 40. Out of 100 patients 73 patients have got CRP value greater than 40 and 27 patients have got CRP value less than 40.



CRP ABOVE 40	OUTCOME	PERCENTAGE
27	9	33.33
27	18	67.66



This table implies the outcome in patients whose crp values are above 40 out of 27 patients whose CRP values greater than 40 9 patients got amputated and 18 patients wound granulated and healed after adequate wound debridement and slough excision.

DISCUSSION

After analyzing the results of our study conducted from January 2012 to November 2012 we observed significant level of correlation between glycemic control CRP level and outcome in terms of wound healing and amputation.

After analyzing CRP levels in all patients it is observed that in patients with CRP level greater than 40 were 27. Out of which 9 got amputated and in the remaining patients the wound healed after surgical treatment.

In our patients out of 100 patients who were evaluated in our study the least fasting value was 127 highest fasting value was 225 similarly patient with least post prandial level is 208 patient with highest post prandial level is 394.

It is observed that persistently elevated fasting and post prandial also had elevated CRP level in the blood, which in term is poor prognosis in terms of outcome.

All the hundred patients were grouped into 5 subsets on increasing level of fasting blood sugar. In which highest number 27 persons(27%) falls in 2^{nd} subset(blood sugar values between 147-166).

22 patients falls in 3rd subset(blood sugar between 167-186).21 patients falls in 1 st subset (blood sugar between 126-146).17 patients falls in 4rth subset (187-206).and 13 patients falls under 5th subset (values >207).

Similarly the same patients were divided into 5 subsets based on rising post- prandial level. Out of which 31 patients(31%) falls in 2nd subset(blood sugar value between 241-280).23 patients falls under 3 rd subset (blood Sugar value between 281-320).18 patients falls in 1 st subset blood sugar value between 200-240. 15 patients falls under 4rth subset blood sugar value between 321-360.13 patients falls in 5th subset blood sugar value greater than 360.

Based on several studies and results so far conducted , it has been clearly stated the glycemic control in terms of fasting and post- prandial plays a pivotal role in the outcome of diabetic foot patients.patients who had persistently elevated fasting and post prandial levels has gone for amputation, our study too concludes the same

That the patients who had persistently high fasting and post prandial level went in for amputation.

So we concluded that, by and far the most important factor influencing the outcome in diabetic foot patients is the glycemic control in terms of outcome amputation and wound healing.

In patients with elevated fasting and post prandial blood sugar in consistently elevated manner the response to treatment was poor with antibiotics and there was no improvement in wagner's grading since the wound healing was low, the time taken for granulation tissue to appear was prolonged, when compared to patients with good glycemic control.

In certain patients the CRP level was also elevated to critical level of 40 according to our study and apparently was treated by amputation.

So, it is highlighted and emphasized that there is a strong correlation between glycemic control and outcome in terms of amputation and wound healing in patients with diabetic foot.

Good glycemic control significantly reduces the morbidity in patients with diabetic foot.

After analyzing the results drawn from 100 patients it has been clearly stated that CRP level assumes significance in relation to outcome in patients with diabetic foot.

Usually the CRP level is not detectable in the blood.values<5 mg is normal.whereas in diabetes whose glycemic status is poor CRP is found to be significantly elevated, invariably in all those patients with diabetic foot.

After analyzing the results drawn by **Lin C W et al** , **Baris A kinci, Sabiullah Amanullah,** it was concluded that values < 45 -50 mg indicates good prognosis in diabetic footin terms of outcome which may be amputation or healing.

In our study out of 100 patients 27 patients has elevated CRP levels > 40 mg .out of these 27 patients 9 patients were amputated either by above and below knee amputation and remaining patients healing of wound was observed.

It is interesting to note that patients who had elevated CRP > 40 also had extensive with gross level of infection when compared to rest of patients.this was assessed using wagner's grading. Which grade the patients into 5 subgroups based on depth of tissue involvement.

Out of 9 patients who went in for amputation 3 patients falls under wagners grade 4 and 6 patients wagners grade 5.

By several studies and research conducted so far, in the medical literature it has been concluded that the patients in higher end of grading has ultimately gone for amputation which was similar to that of results drawn in our study.

Out of 27% of patients with elevated CRP level >40, 9% of total patients went in for amputation and in 18 patients wound healed. so the chance of having the risk of amputation when the CRP level is greater than 40 is 33.33%.

It is also observed from our study that the patients have got elevated CRP but not went in for amputation have sustained slower rate of wound healing, whereas in those patients who have got CRP < 40 had good response to treatment with quick recovery and wound healing.

CONCLUSION

From our study it is concluded that glycemic control and CRP has definitive correlation with outcome which is proved by patients with persistently elevated blood sugar levels and CRP went in for amputation in our study.

It is also concluded that 33.33% (9 patients out of 27) patients with elevated CRP above the critical value of 40 have went in for amputation. Rest of the patients with CRP < 40 went in for wound healing with appropriate surgical management.

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Lawrence A.Lavery, DPM, MPH,J. Christopher Moore, DPM, John M. Schuberth, DPM,Dane K. Wukich, MD,11 Andersen, MD,12and John V. Vanore, DPM

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Yutaka Takata Kazuo Sonoki Kiyoshi Fujisawa Toshihiro

AnsaiSumioAkifusa

Koji FujiiMitsuo Iida3 and TadamichiTakehara

NAME	AGE	SEX	IP NO	FASTING
Manogar	52	m	44200	145
				135
				127
Mogli	55	m	14782	147
				139
				98
jayakumar	67	m	11335	170
				148
				78
Rajeshwari	38	f	30544	174

				135
				107
Kamatchi	40	f	10108	181
				137
				87
Jayachandran	55	m	42613	186
				106
Sagayamary	45	f	44278	187
				148
				116
Krishnamma	65	f	50682	194
				167

				123
Muthu	36	m	32060	194
				156
				102
Sudhamani	70	f	46187	195
				163
Sarroja	70	f	50765	197
				178
				124
Puspha	65	f	17276	199
				168
				132
Lucas	80	m	27954	201

				156
				112
Mani	65	m	44672	202
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				123

Muthuraj	40	m	44194	208
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				128
Rammaya	70	m	52100	209
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Dharanidharan	35	m	6988	211
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meenakshi	53	f	23312	213
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Krishnaveni	60	f	51941	217
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Selvaraj	63	m	26309	219
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Udhayakumar	45	m	42537	219
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Jothi	58	f	42336	224
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Chandrasekar	30	m	27854	225
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				156
durai	72	m	74521	185
				163
				144
Baskar	48	m	4359	137
				124
Munusamy	55	m	41567	183

				146
				98

POST PRANDIAL	C REACTIVE PROTEIN	GRADE
243	45.6	grade 2
245		
220	36.5	
244	45.6	grade 2
219		
175	23.9	
289	48.1	grade 2
203		
167	40.2	
296	41.2	grade 2

242		
195	29.7	
307	45.1	grade 2
256		
157	38.2	
315	42.9	grade 2
211		
316	45.9	grade 3
239		
175	40.1	
330	67.2	grade 3
290		

198	42.3	
332	57.2	grade 3
279		
203	41.2	
335	40.3	grade 3
254		
342	48.3	grade 3
256		
214	26.9	
345	52.4	grade 3
283		
180	34.9	
349	73.2	grade 2

245		
187	37.6	
353	96.3	grade 3
254		
187	45.4	

361	78.2	grade 4
271		
207	59.8	
364	69.2	grade 4
321		
354	70.3	
367	76.3	grade 5
304		
264	49.3	
371	87.9	grade 5
267		
287	76.3	

377	65.2	grade 5
339		
285	63.6	
238	56.2	
380	59.4	grade 5
287		
246	55.3	
313	62.3	
382	42.2	grade 3
311		
275	43.2	
386	123.4	grade 5
312		

274	87.3	
316	85.7	grade 5
184		
156	54.3	
394	67.8	grade 4
345		
254	69.3	
312	53.9	grade 3
287		
243	41.7	
232	57.4	grade 1
176		
308	61.3	grade 3

267		
205	47.3	

OUTCOME
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healed
healed
healed


healed
healed
healed
healed


healed
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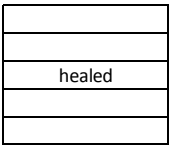
healed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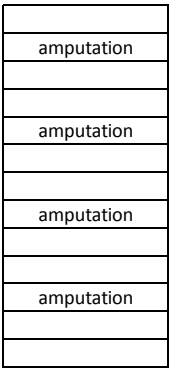
amputation
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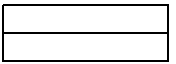

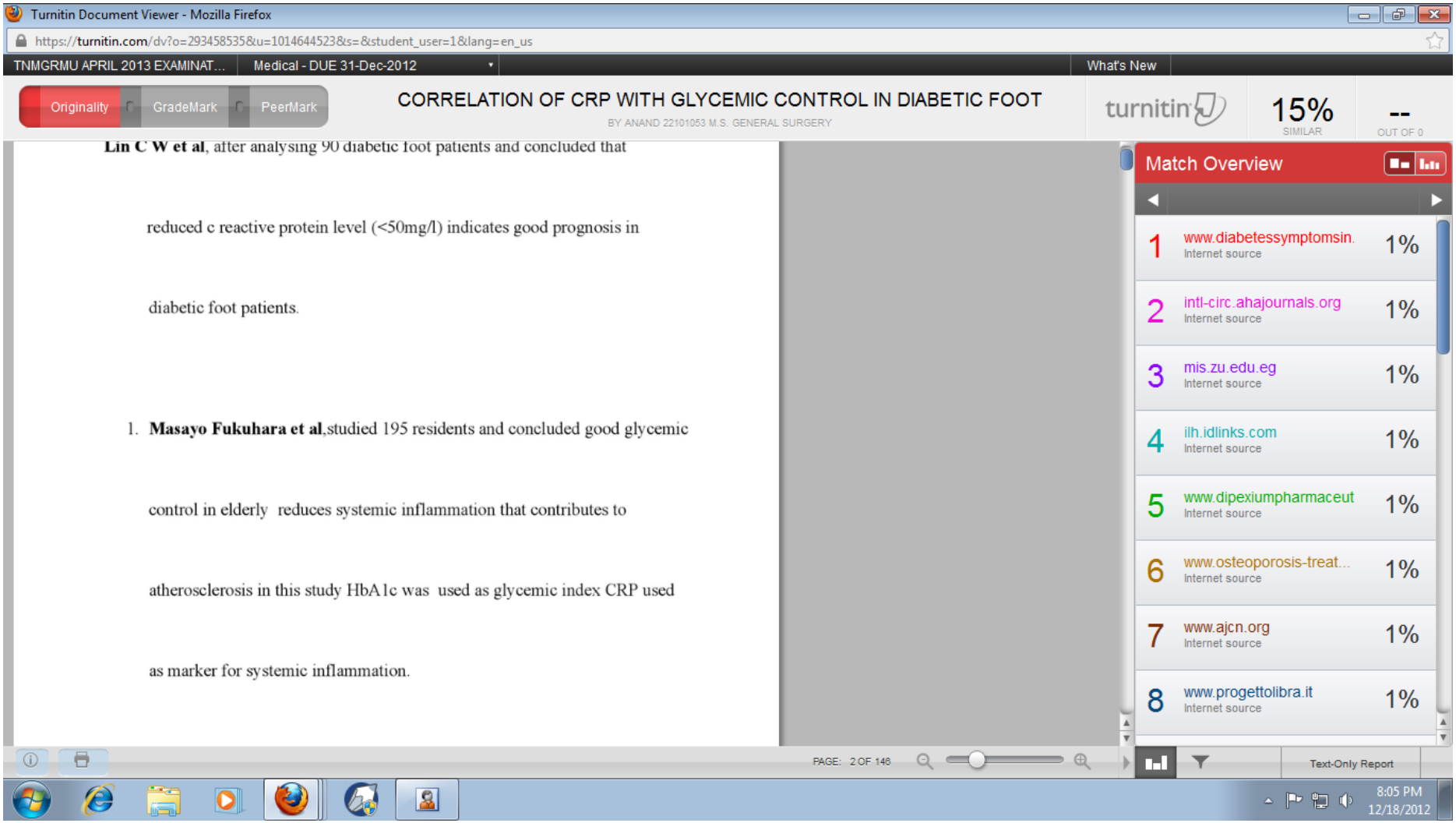
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